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| **CESTODES/**  **TAPEWORMS:** | ***Taenia solium &***  ***Taenia saginataas* adults**  **INTENSTINAL** | ***Diphyllobothrium latum***  **INTESTINAL** | ***Dipylidium caninum***  **INTESTINAL** |
| **English Name** | *T. solium* = pork tapeworm  *T. saginata* = beef tapeworm | Fish Tapeworm | Dipylidiasis |
| **Mode of transmission and the infective form** | Eating muscle tissue infected with larval cystercerci | Improperly cooked fish with larval stage; Pleurocercoid larvae | Eating infected flea |
| **Life Cycle** | * Indirect   + Cow eats shit/proglottids in grass; larvae get into its muscle and form cystercerci; we eat the cow; adult matures in our GI; we shit out gravid proglottids | * Indirect * We eat fish with larvae in them; adult forms in our gut; we shit out proglottids/eggs | * Indirect   + Humans get infected by ingesting fleas infected with cysticercoid (larvae) |
| **Migration w/in Humans** | * Ingested cystercerci mature into adults that hook onto the intestine via a “scolex” | * Larva matures into tapeworm in small intestine; Takes about 3 months * Can grow 10m long (**largest human tapeworm)** | * Scolex attaches in the intestines * Adults grow in small intestines * Gravid proglottids are pass in feces or emerge from perianal area and are taken up by larval stage of flea |
| **How is pathology produced?** | * Virtually no clinical effect. * Abdominal discomfort * The main symptom is often the passage of proglottids trough the intestinal tract. * Migrating proglottids→ **Appendicitis or cholangitis** * *T. solium* taeniasis is less frequently symptomatic than *T. saginata* taeniasis. * The most important feature of *Taenia solium* taeniasis is the risk of development of cysticercosis. | * Virtually no clinical effect * Epigastric pain, nausea, vomiting * Occasional intestinal obstruction, diarrhea, and abdominal pain * Occasionally (<1% of time) causes Low levels of B12-> megaloblastic anemia--from vitamin B12 uptake by strobila | * Virtually no clinical effect * Abdominal discomfort, anal pruritus, and diarrhea |
| **Diagnosis –**  What sample?  What is seen? | * Eggs in feces * C:\Users\ccortes\Desktop\kabisa_1219.jpgTaenia_eggA * Only proglottids can differentiate b/w beef/pork * + Scolex: rarely found   + Left beef—suckers only   + Right pork—hooks and suckers * + Gravid Proglottids-uterine branches injected with indian ink—more in T. saginata (picture on the R) | * Proglottids/eggs in feces * C:\Users\ccortes\Desktop\1355.JPGC:\Users\ccortes\Desktop\dlatum_egg.jpg * Operculated Eggs with knob on the bottom of the shell or proglottids in feces | * Eggs (colorless egg packets) or proglottids in feces * C:\Users\ccortes\Desktop\Dipylidium_caninum_ovum_1.jpgC:\Users\ccortes\Desktop\dipy5.jpg |
| **Definitive host** | Humans--intestines | humans, bears, minks & other animals (Any fish eating carnivore) | Pet-owners (Canids and felids) and **children** |
| **Intermediate host(s)** | *T. solium* = pigs or human (*see cystercercosis*)  *T. saginata* = ox (ie, eating larvae OR eggs of pork tapeworm infects us; for beef tapeworm only larvae infect us), cattle larval form (cysticercus) in muscle | larval stages in water, picked up by water flea (Copepod 🡪crustacean), then by fish (source of human infection) | fleas |
| **Form transmitted from human to next host** | Cattle must ingest grass contaminated with human feces. Humans do not get beef/pork tapeworm from ingesting eggs, only by ingesting the larval (cysticeri) form (in meat). | Eggs hatch in water and are ingested by water flea, which are then eaten by fish | Fleas ingest gravid proglottids that are passed intact in the feces or emerge from perianial region of either animal or human |
| **Geographical foci** | Both: Worldwide  *T. solium* = more prevalent in pork-eating countries and poorer communities where humans live in close contact with pigs and eat undercooked pork. | Worldwide | Worldwide |
| **Treatment/Prevention** | * Prazyquantel   Prevention   * Cooking “well done”/deep freezing food + meat inspection * Hygiene (cows and pigs must eat human shit to get infected!) | * Niclosamide * Prazyquantel | * Prazyquantel |

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| **CESTODES/**  **TAPEWORMS:** | ***Taenia solium* larval disease**  **TISSUE INFECTION** | *Echinococcus granulosus* **TISSUE INFECTION** |
| **English Name** | Cystercercosis | Hydatid Cyst |
| **Mode of transmission and the infective form** | Ingestion of eggs from *human* feces (eggs are formed by adult worms living in human intestines) | Ingestion of embryonated eggs (not proglottid) |
| * **Life Cycle** | * Direct | * Indirect * We eat dog shit; get cysts 🡪 no adults formed in humans (only in dogs)   Note: *E. multilocularis* is emerging problem.. rodents/ foxes.. extensive cysts |
| * **Migration w/in Humans** | * Gastric acid and pancreatic enzymes release oncosphores * Oncosphores penetrate intestinal wall and are disseminated with the blood * Once distributed to peripheral tissues they die, swell, and calcify, as they cannot complete their life cycle | * Oncosphores (motile larvae) hatch and penetrate intestine * Enter circulation and form hydatid cysts in liver, lungs, etc. |
| * **How is pathology produced?** | * **The most frequently reported tissue locations** are skin, skeletal muscle -> Do not cause symptoms. * Asymptomatic subQ nodules & calcified intramuscular nodules can be encountered. * Death can occur suddenly. * Extracerebral cysticercosis can cause ocular, cardiac, or spinal lesions with associated symptoms. * Cerebral cysticercosis or **neurocysticercosis** → seizures, mental probs, focal neurologic deficits, & signs of space-occupying intracerebral lesions * Cysticerci form in muscles & organs including the brain * Very problematic for brain as cestodes die/swell (can take years) | * Silent for years * Hepatic involvement: abd pain, a mass in the hepatic area, & biliary duct obstruction. * Pulmonary involvement🡪chest pain, cough, and hemoptysis. * Rupture of the cysts can produce fever, urticaria, eosinophilia, and anaphylactic shock, as well as cyst dissemination. * Other organs may be affected (brain, bone, heart) |
| **Diagnosis –**  What sample?   * What is seen? | * Antibody detection   + Immonoblot and enzyme immunoassays (EIA) * Bx of the affected area * ↑ IgE & Eosinophils * CT scan, MRI scan, or x-rays to detect lesion * Macintosh HD:Users:elisafuray:Desktop:Screen Shot 2012-11-06 at 2.00.08 PM.png * “Rice grain” calcifications in skeletal muscle | * **Antibody detection**    + Indirect hemagglutination (IHA), indirect fluorescent Ab (IFA) tests, & enzyme immunoassays (EIA) * Hydatid Cyst. Ultrasonography and/or other imaging techniques supported by positive serologic tests * hydatid_cyst4 * In seronegative patients with hepatic image findings compatible with echinococcosis:   + Ultrasound-guided fine needle biopsy may be useful for confirmation of diagnosis;   + Precautions to control allergic reactions or prevent secondary recurrence in the event of leakage of hydatid fluid or protoscolices.   + C:\Users\ccortes\Desktop\Egranulosis_protoscoleces_PHXChild_B.jpgProtoscoleces in a hydatid cyst |
| **Definitive host** | Humans | Small intestines of dogs and relatives |
| **Intermediate host(s)** | Humans  cysticerci in muscle + organs= cysticercosis 🡪develop after eating eggs from human feces  Note this disease also occurs in pigs, from eating human shit | Many animals (especially humans—causing hyatid dz) & livestock—sheep, goat swine) |
| **Form transmitted from human to next host** | Eggs (between humans or autoinfection) | Release of cysts or protoscoleces |
| * **Geographical foci** | * Worldwide distribution (pork-eating countries) * Neurocysticercosis may account for at least 10% of seizures seen in some ED & >2% of neurological/ neurosurgical admissions. * Most persons who die from cysticercosis in the US are foreign born. | Worldwide  frequently in sheep-raising countries, incl U.S. (rural, grazing areas🡪dogs ingest organs from infected animals) |
| **Treatment/Prevention** | **Surgery:**   * Remove Cysticerci (in Eye, ppl not responsive to drug treatment, or to reduce brain edema)   **Neurocysticercosis**   * **Sx tx** w/ anti-seizure meds.   + Antiparasite drugs (**Albendazole** or **Mebendazole**) & anti-inflammatory (corticoids) drugs & anticonvulsant (when needed). * **Asymptomatic**. Untx larval infections often subside (symptom-wise) within 2 to 5 years   Prevention   * Hygiene (only way to get this dz is to eat human poop) * Tx ppl harboring adult T. solium (to ↓ transmission) | **Surgery**:   * Removal of the parasite mass—not 100% effective.   Danger from ruptured cyst:   * release of Ag→shock * release of protoscolices🡪 new cysts * After surgery, medication may be necessary to keep the cyst from recurring. * **Albendazole** * **Mebendazole** |

* **Helminths**
  + ENDOPARASITES
    - PLATYHELMINTHS (FLAT worms—PLAT is FLAT)
      * **Cestodes (tapeworms)**
        + parasitic; may be meters long
        + tegument (on surface); absorbs nutrients from host thru skin
        + **long, ribbon/segmented**
        + head = scolex with hooks, rostellum, suckers

scolex—anterior attachment organ

* + - * + neck – proglottids, (extension of body that form strobili)
        + Stobili--string of proglottids

immature near neck

mature--gravid w/ fertile eggs

* + - * + hermaphrodites
        + no body cavity, no digestive tract
        + Adults found in intestine – virtually NO clinical effect
        + Larvae hatch from eggs, eaten by intermediate host – larvae penetrate thru intestinal wall into tissues = **most damaging dz by larvae**
        + Diagnostic: eggs/proglottids in feces
        + Examples

Intestinal (transmitted by ingestion)

T. solium

T. sajinata

Diphilobothrium latum

Dipilidium caninum

Tissue

T. solium-cysticercosis

Transmitted by ingestion

E. granulosus

* + - * Trematodes (Flukes)
        + Schistosomiasis
    - NEMATHELMINTHES (ROUND worms)
      * Nematodes
        + Lympatic filariasis
        + Hookworm
        + Trichuriasis
        + Ascariasis

1 in 5 ppl on Earth have this

* + - * + Onchocerciasis